Early Systolic Sounds In Aortic Valve Stenosis

G. L. DONNELLY* AND E. A. VANDENBERG

From the Thoracic Unit and the Unit of Clinical Investigation, Royal North Shore Hospital, Sydney, Australia

The association of early systolic sounds with aortic valve disease has long been recognized (Potain, 1900; Oriás and Braun-Menéndez, 1939). The genesis of this sound and indeed the timing in relation to aortic valve opening has been disputed. Some authors (Wolferth and Margolies, 1945; Wiggers, 1949; Minhas and Gasul, 1959) suggest that they occur at the time of aortic valve opening, while others (Leatham and Vogelpoel, 1954; Reinhold, Rudhe, and Bonham-Carter, 1955) postulate an origin in the aortic wall during the systolic ejection period. This has led to these sounds being variously termed early systolic clicks, and ejection sounds.

For these reasons, a study was carried out in a group of patients with suspected aortic stenosis. The purpose of this study was to clarify the relationships between these sounds and left-sided pressure events, to assess the frequency and significance of such sounds, and to make observations relevant to their mechanism of production.

SUBJECTS AND METHODS

Studies were carried out on 24 patients with aortic valve stenosis. In 4 patients a significant degree of aortic incompetence demonstrated by retrograde aortography was present, this being the dominant abnormality in one. In 9 other patients, short early diastolic murmurs were present but were not accompanied by other clinical evidence of aortic incompetence.

There were 18 male and 6 female patients, the ages ranging from 11 to 62 years; 6 were less than 22 years and were presumed to have congenital lesions. In 5 of these, withdrawal tracings or angiographic studies showed the stenosis to be at valve level. The remaining 18 patients were 39 years or more, and in this group the ætiology was uncertain.

External phonocardiograms were recorded by an N.E.P.† phonocardiographic system with a crystal microphone, using medium frequency and additionally

in some cases low frequency recordings (Leatham, 1952). Recordings were made at the site of maximal loudness of the early systolic sound if audible, or at the left sternal border in the fourth intercostal space. The amplitude of the vibrations thus recorded was used to compare the intensity of the different sounds or groups of vibrations.

Left ventricular and arterial pressures were measured simultaneously, using equisensitive Statham P23G pressure transducers with carrier wave amplification. Zero reference was at the sternal angle. In 21 patients, left ventricular pressure was obtained by direct percutaneous puncture, using a 10 cm. 18 S.W.G. needle connected by nylon tubing (length 45 cm. internal diameter 1 mm.) to the transducer. In 2 patients, percutaneous retrograde femoral catheterization was employed, and in one transseptal puncture, the catheters being similarly connected to the recording system. Arterial pressures were recorded in most instances from the brachial artery via a short indwelling polythene cannula, and in 3 patients from the ascending aorta immediately above the aortic valve via a retrograde femoral catheter.

Aortic valve opening was assessed as the time when left ventricular pressure exceeded arterial diastolic pressure. The use of brachial diastolic pressure to assess the time of aortic valve opening introduces a possible timing error as central aortic diastolic pressure may be slightly higher in some cases, particularly if incompetence is present. This error was measured in 6 patients from simultaneously recorded pressures in the brachial artery and the ascending aorta. At the operative rates of left ventricular pressure rise of 1600 to 4600 mm. Hg/sec., the use of brachial artery pressure in these patients resulted in a slightly premature estimate of aortic valve opening (range 0–6 msec.; mean 1·8 msec.). No correction for this possible error in timing has been made, the error being within the range of measurement error. The transmission delay of the left ventricular pressure pulse recording system relative to the phonocardiogram was tested externally and found to be 5 msec. for the needle probe and 10 msec. for either catheter recording system. The data presented below have been corrected for these delays.

Recordings were made on a 6-channel N.E.P. ultraviolet recorder employing optical galvanometers at a paper speed of 80 mm. per second. A simultaneous time base of 100 c.p.s. was used to facilitate measurements and obviate recorder error, with additional time identification

lines at 200 msec. intervals. Measurement was thus possible to the nearest 5 msec.

Time intervals were measured to the nearest 5 msec. from the onset of the first major vibrations of the first heart sound to the onset of major vibrations of the additional early systolic sound(s), and from the onset of the first major vibrations of the first heart sound to aortic valve opening in the same beat. All patients were in sinus rhythm and three consecutive regular beats were analysed in each patient. Maximal variation in these and in other parts of the record was 5 msec., excluding extrasystolic and post-extrasystolic beats. Mean values were estimated to the nearest 5 msec.

RESULTS

Of 24 patients studied, 4 had no early systolic sounds recorded either clinically or on phonocardiography. All were men with calcific aortic stenosis. In 2 further patients, sound vibrations continued from the onset of ventricular contraction into the ejection period, but it was not possible to identify separate groups of vibrations. In the remaining 18 patients, one or two additional groups of rapid vibrations could be separately defined from the initial major vibrations of the first heart sound (Fig. 1 and 2).

In seven recordings two separate groups of vibrations could be identified. In each case the initial group of vibrations occurred at or slightly before aortic valve opening and the latter group of vibrations followed valve opening (Fig. 2). In 11 cases, a single early systolic sound was recorded.

This sound occurred at or slightly before the time of valve opening in 10 patients and after valve opening in one. Fig. 3 shows the time relationships of the recorded early systolic sounds to valve opening. The time interval from the onset of the QRS deflection to aortic valve opening (Q-AVO) has been plotted against the interval from the onset of QRS deflection to the onset of added sound (Q–C). The added sounds can be seen to fall into two groups, the first group occurring in the isovolumetric period before valve opening and the second group occurring in the early ejection period. For purposes of discussion, the earlier type, including two timed to occur at valve opening, will be designated C_1 and that in the ejection period will be termed C_2.

The basic characteristics of both types are outlined in the Table. An isovolumetric sound (C_1) occurred in 17 patients. The onset of the sound was 0 to 20 msec. before aortic valve opening and 20 to 60 msec. after the onset of the first sound. C_1 was thus more constantly related to aortic valve opening than to the initial vibrations of the first heart sound. In addition, C_1 was the dominant sound recorded in 9 of these 17 patients and was equally dominant in 3. These 12 patients included 3 of the 6 who were 21 years and younger, 4 of 6 women and 5 of 12 men. However, of those patients with peak systolic gradients in excess of 60 mm. Hg, C_1 was dominant in all 3 who were 21 years and younger, in 3 of the 4 women but in only 2 of 6 men.
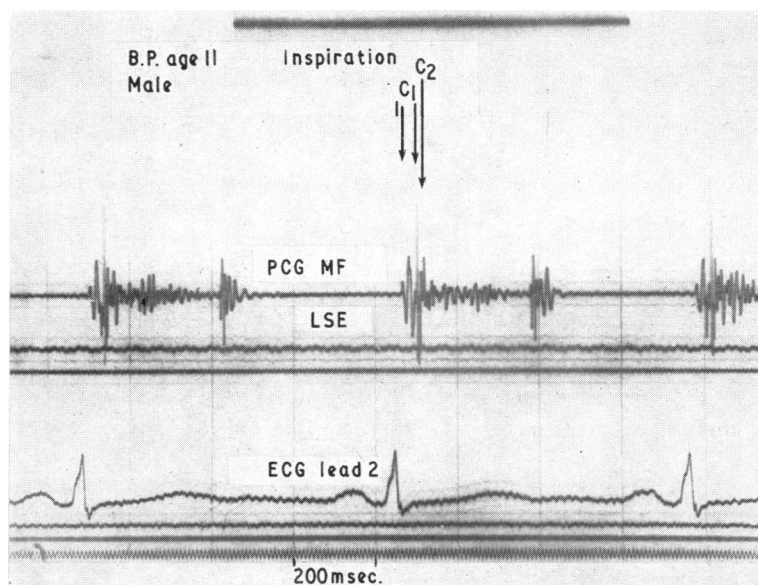

FIG. 1.—Phonocardiogram recorded at the left sternal edge in an 11-year-old boy with moderately severe aortic valve stenosis. Two additional components (C_1 and C_2) follow the initial major vibration (1) of the first heart sound.
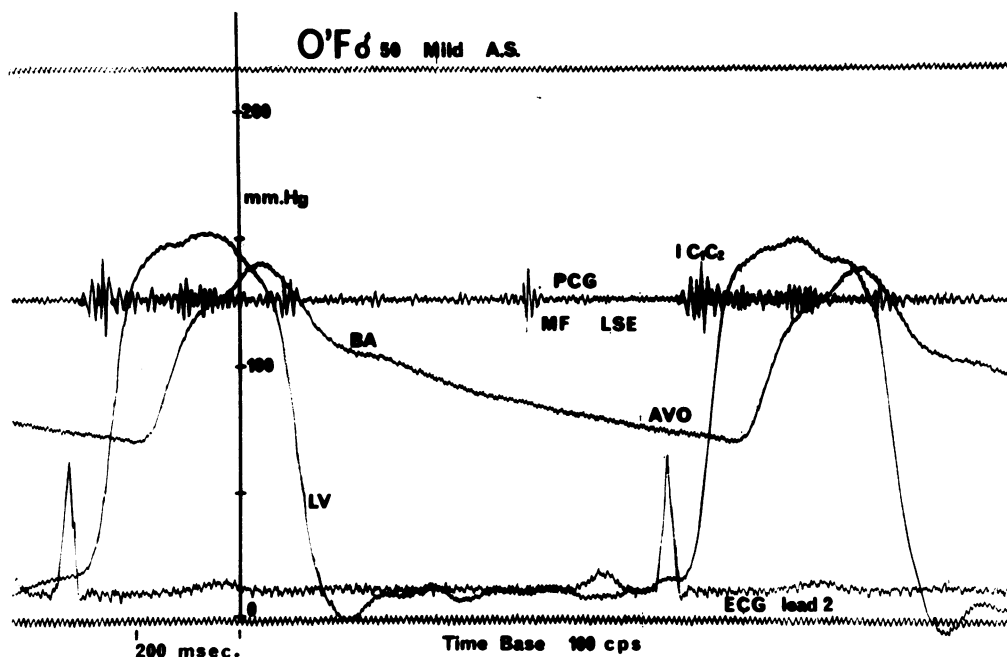
FIG. 2.—Simultaneous pressure and phonocardiographic tracings in a man aged 50 with mild aortic stenosis, showing C_1 preceding aortic valve opening and C_2 following valve opening.

An ejection sound (C_2) was discernible in 8 records but was seldom of marked intensity. The onset of C_2 was from 10 to 35 msec. after aortic valve opening and from 45 to 85 msec. after the onset of the first heart sound. C_2 was the dominant sound in only three instances and was equally domi-nant in one. These four were all patients with mild aortic stenosis, two being clearly congenital. A clearly defined prominent C_2 was not a feature of more marked degrees of aortic stenosis.

DISCUSSION

Earlier writers have disputed the timing and source of these sounds. Wolferth and Margolies (1945) attributed the early systolic sound in aortic valve disease and hypertension to aortic valve opening, and this concept was supported by others (Wiggers, 1949; Minhas and Gasul, 1959). Leatham and Vogelpoel (1954) recorded early systolic sounds in a small series of patients with aortic stenosis, aortic sclerosis, and coarctation of the aorta. On the basis of indirect carotid pulse tracings they felt that these sounds were ejection in timing. This method is unreliable for the precise timing of aortic valve opening (Wiggers, 1949; Minhas and Gasul, 1959). A slight rise in aortic pressure may occur before valve opening (Fig. 4), as has been pointed out by Wiggers (1949).

In the present study true ejection sounds were less common than the earlier isovolumetric sound. This isovolumetric sound is closely related to aortic valve opening over considerable variation in the isovolumetric period. In most cases, however, it preceded valve opening by a small but measurable interval.
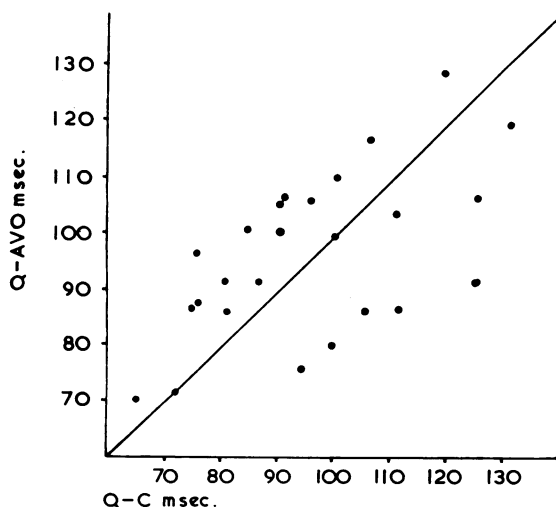

FIG. 3.—Graphic correlation of the additional components with aortic valve opening. Q-AVO=interval from onset of QRS to aortic valve opening. Q-C=interval from onset of QRS to onset of the early systolic sound.

TABLE

Occurrence	Relative intensity*	Time relationship to 1st sound (msec.)	Time relationship to aortic valve opening (msec.)
C_1 17 out of 24 (71%)	Dominant in 9 Equal to H_1* in 1 Equal to C_2 in 2	+20 to +60 (mean +35)	−20 to 0 (mean −10)
C_2 8 out of 24 (33%)	Dominant in 3 Equal to H_1* in 1	+45 to +85 (mean +60)	+10 to +35 (mean +20)

* First heart sound (H_1) dominant in 12.
Intensity based on recordings at medium frequency (MF) at site of maximal intensity of early systolic sound.

In a recent study of the first heart sound in the dog, Shah *et al.* (1963) have defined a similar group of vibrations, designated as the second component of the first heart sound. They have shown that this component occurs during the isovolumetric period and is maximal in the left and seldom seen in the right intracardiac phonocardiogram. They also concluded that its magnitude was related to the maximal rate of pressure rise in the left ventricle. The latter occurred synchronously with the second component. Although in the present study it has not been possible to measure continuously the instantaneous rates of pressure rise, the timing of the isovolumetric component in aortic valve disease

and of the normal second component are such as to suggest a common identity. The accentuation of this component would not be unexpected in aortic stenosis and in systemic hypertension if it were related to the maximal rate of pressure rise in the left ventricle, as this is considerably increased in these conditions (Gleason and Braunwald, 1962).

Luisada and Shah (1963) have recently emphasized the fact that close splitting of the first sound with audible first and second components is a normal phenomenon. Leatham (1954) found that the separation of these two components was 20 to 30 msec. in normal people, using medium or high frequency recording. More recently, Luisada and
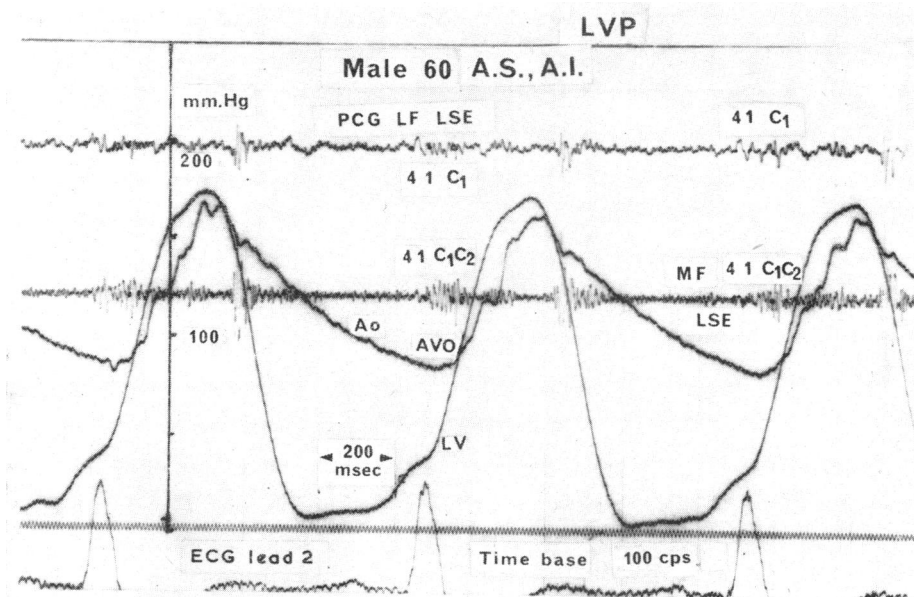

FIG. 4.—Tracings from a patient with dominant aortic incompetence and a slowly rising ventricular systolic pressure curve. Central aortic pressure begins to rise before aortic valve opening. The 1-C_1 interval is quite prolonged because of the slow pressure ascent.

di Bartolo (1961) using selective high frequency (500 to 1000 c.p.s.) have found this interval to be 30 to 40 msec. In the present series, the isovolumetric sound occurred from 20 to 60 msec. after the onset of the first sound. This time delay is essentially dependent on the duration of the isovolumetric period. In many patients, however, a delayed first sound may be present due to raised atrial and left ventricular end-diastolic pressure, with resultant shortening of this interval. In addition, with severe aortic stenosis, the rapid rate of pressure rise in the left ventricle would have a similar effect.

The true ejection type of sound is less common. However, in some patients it may be partly obscured in the onset of the systolic ejection murmur. Although identified in half the patients, it was probably sufficiently loud to be audible in only four, all with mild aortic stenosis. It has been observed in pulmonary valve stenosis that pulmonary ejection sounds indicate mild degrees of obstruction and exclude more significant stenosis (Leatham and Vogelpoel, 1954). Thus, the situation is similar in aortic valve stenosis.

In this study true ejection sounds have been found 10 to 35 msec. after valve opening. Shah *et al.* (1963) found the third or ejection component of the first heart sound in dogs to occur 10 to 31 msec. after the onset of pressure rise in the aorta. It appears likely, then, that both types of early systolic sounds are merely augmented normally occurring components of the first heart sound.

The auscultatory differentiation of each type is sometimes uncertain, though the isovolumetric type resembles a "close split" and the later or ejection type a "wide split". The former is generally heard better at the apex than over the aortic area and seems to be less clicking in quality. An augmented third component or ejection sound is not well heard with loud systolic murmurs. Phonocardiography may help by a more precise measurement of the degree of "splitting". Our results indicate that a separation from the first component of more than 50 msec. indicates the sound to be either a third (ejection) component or a second component delayed sufficiently to exclude significant stenosis. A loud dominant second component does not, however, indicate surgically significant stenosis, even in older patients.

Absence of the second component on the phonocardiogram was noted in only four patients in this study. All of these were men with severe calcific aortic stenosis. It is thus possible that some flexibility of the aortic valve is necessary for the production of this sound, in much the same manner as is suggested for the opening snap of the mitral valve in mitral stenosis. A loud isovolumetric sound was recorded in one man with severe calcific aortic stenosis (Fig. 5).

Much of the controversy regarding early systolic

FIG. 5.—A loud C_1 present in a man aged 58 with severe calcific aortic stenosis.

sounds in aortic stenosis appears to be due to confusion of these two types of early systolic sounds. It may be argued that an isovolumetric sound is simply a normal or slightly increased degree of "splitting". This sound, however, is undoubtedly abnormally loud and readily interpreted as an added sound in many patients with aortic stenosis, as compared to normal subjects. In the present series, the clinical impression of an early systolic sound was more likely to be associated with an isovolumetric sound than an ejection sound.

The terminology of these sounds needs clarification. A sound that is labelled ejection must be widely separate from the first sound. When loud and easily audible, it indicates hæmodynamically insignificant stenosis. Significant stenosis is usually associated with a "splitting" of the first sound in the absence of marked calcification. The term early systolic click is confusing and probably should be abandoned. Lewis (1962), summarizing current concepts, suggests that the term systolic click be reserved for mid or late systolic sounds and that the term ejection sound be used for sounds occurring in the ejection period.

SUMMARY

In a series of patients with aortic valve stenosis, and in some cases additional aortic incompetence, two types of early systolic sounds were found.

The commoner type of early systolic sound occurred at or just before aortic valve opening and was probably an accentuated second component of the first heart sound. A true ejection sound was less frequent and when dominant was not associated with significant aortic valve obstruction. Both sounds appeared to be more common in the younger patients in whom the valve stenosis was presumably congenital and not calcific in type.

Differentiation of the two types is clinically possible but in some cases is uncertain, even with phonocardiography and a simultaneous electrocardiogram.

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